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MANIFESTED IN THE LARYNX.

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HONORARY PROFESSOR OF LARYNGOLOGY, AND LECTURER ON DISEASES OF THE THROAT AND CHEST IN JEFFERSON MEDICAL COLLEGE, PHILADELPHIA.

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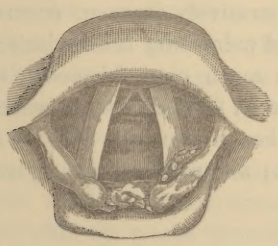
Tuberculosis as manifested in the larynx, includes, as discussed in this paper, the entire range of pathological changes which ensue in the larynx as a result of its infiltration by tubercle. Attention will be directed both to the clinical pathology of the malady as revealed by laryngoscopy, and to its histological pathology as revealed by microscopy.

Following the resort to laryngoscopy as a means of objective diagnosis, announcements were soon made that the early presence of miliary tubercle could be detected in the mucous membrane of the living larynx; and that the entire progress of the tuberculous process could be studied from time to time in the laryngoscopic image. Similar assertions, indeed, are still made.

Small globular or semiglobular nodules, pin-head or thereabout in size, yellowish in tint, seen isolated or clustered at different portions of the laryngeal mucous membrane, were attributed to accumulations of miliary tubercle.

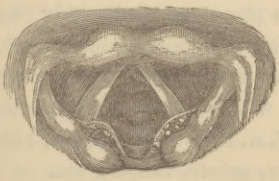
The customary destructive metamorphosis of these bodies, long before the death of the patient, prevents verification or denial of their initial tuberculous character upon positive premises. By prolonged observation, however, it has become demonstrated that these tuberculous-looking nodules (Figs. 1 and 2) always occupy localities normally beset with mucous

Fig. 1.



Distended mucous glands.

Fig. 2.



Distended mucous glands (of three years' standing).

[1] A paper read before the Pathological Society of Philadelphia, Sept. 28, 1882.

glands;[1] whence the inference has arisen that they are not tubercles; but are rather hypertrophically distended mucous glands, filled, by occlusion of the orifices of their ducts, with accumulated products of secretion and desquamation; inflamed, perhaps, by some specially irritative quality in the hyper-secretions of the coexisting chronic catarrhal laryngitis. The ultimate destruction of these glands results chiefly from necrotic inflammatory processes set up by the pressure of tuberculous infiltrations around them and between their individual acini. In this manner follicular ulcerations are produced whose racemose configuration so closely resembles the crenated margins of some tuberculous ulcerations, as to render it often impossible to distinguish one from the other, save under the lens of the microscope.

Nodules, similar in their gross laryngoscopic aspect to those which have just been mentioned, sometimes remain unchanged for many months. Several examples have occurred in my own practice; the appearance depicted in the second illustration (Fig. 2) having lasted, to my knowledge, for more than three years, in the larynx of a practising attorney of this city. These certainly cannot be tubercles, except under the questionable hypothesis of their calcification.

The long-mooted question of the existence of tubercle in the larynx, seems to have been set at rest in the affirmative; and chiefly by quite recent researches of Heinze[2] and Eppinger.[3]

Primary Tubercle.—Pathologists acknowledge the possibility of primary infiltration of the larynx with tubercle; but they await satisfactory confirmation of the hypothesis. No record exists, to my knowledge, of detection of tubercle in the larynx of the dead subject, without abundant coexisting tubercle in the lungs. Clinical evidence of such primary deposit is presumptive rather than demonstrative. This presumptive evidence is based solely upon laryngoscopic inspection, which, in individuals in whom no physical signs of pulmonary lesion can be detected, reveals a condition of the larynx known to be more or less characteristic of tuberculous processes in that structure.

Secondary Tubercle.—Secondary infiltration of tubercle in the larynx is generally acknowledged to be of comparatively frequent occurrence. It takes place, as a rule, only in subjects of pulmonary tuberculosis; and, as far as my own records teach, appears much more frequently in the inherited than in the acquired variety. It is, furthermore, associated, as a rule, with secondary tuberculosis in other structures: both at a distance, *i. e.*, intestines, spleen, kidneys, etc.; and contiguous, *i. e.*, trachea, pharynx, palate, tongue, etc.

[1] Inner surfaces of the arytenoid and supra-arytenoid cartilages, lower and inner surface of epiglottis, meso-arytenoid fold.

[2] Die Kehlkopfswindsucht, nach Untersuchungen im pathologischen Institute der Universität. Leipzig, 1879.

[3] Pathologische Anatomie des Larynx und der Trachea. Berlin, 1880.

An *acute tuberculous sore throat* has been described, with considerable detail, by Isambert, Frœnkel, and a few others. It is an acute miliary tuberculosis of the pharynx and larynx, which rapidly ulcerates, and terminates fatally in a few weeks, under further progress as acute tuberculosis of the lungs. Abundant disseminations of confluent patches of miliary tubercle have been observed beneath the epithelium, which bleeds freely when touched. These appear first upon the palate, anterior palatine folds, the tonsils, and the pharynx; and, at a later stage, upon the epiglottis and the larynx. They are exceedingly painful, so much so that deglutition is sometimes impracticable. Ulceration soon ensues, enucleating a certain number of the tubercles; and leaving empty sacs, with more or less deep losses of substance. Death occurs, usually, before extensive ravages can be produced.

Of this affection I know almost nothing personally. One example presented at the Throat Clinic of Jefferson Medical College Hospital, a few years ago, in the person of a lad, whom I had but the one opportunity of examining; and who, as I learned upon inquiry, died a few weeks afterwards.

In 1868, the larynx, from what I strongly suspect to have been a case of this kind, was presented to this Society, by Dr. Tyson,[1] who called attention to the fact that the ulceration began in the fauces, and that the patient, a man, 49 years of age, whom he had seen in consultation, suffered with painful deglutition to an extreme degree. The rapid progress of the disease in this instance, the intense pain on deglutition, the early ulceration in the throat, and the slight amount of laryngeal ulceration found post-mortem, tally very closely with the pathological history of the cases discriminated of late years as examples of acute tuberculosis of the throat.

Presumptive Primary Tuberculosis.—My entire practice has furnished me with but three personal examples of even presumptive primary tuberculosis of the larynx. In two instances it was impossible to detect evidences of pneumonic lesions for several weeks following recognition of the tuberculous larynx. The subjects were all males; aged, respectively, 29, 27, and 21 years.

In two cases, one a driver of an ice-wagon, and the other a sailor, the immediate advent of the lesion was directly attributable to severe cold; probably acute laryngitis, from extreme exposure. The third patient, a miller, had no recollection of having caught cold. Hereditary influence was denied in each case.

In the sailor, pneumonic symptoms first became discernible six weeks after the manifestation of disease in the larynx; and death occurred by apnœa within ten weeks thereafter.

In the driver of the ice-wagon, pneumonic symptoms first became discernible eighteen weeks after the manifestation of disease in the larynx;

[1] Trans. Path. Soc., Phila., vol. iii. p. 74.

and death ensued eight weeks later. In the miller, the first pneumonic symptoms became discernible fourteen weeks (April 3, 1882) after the disease had begun; and at last accounts he was reported as far gone in pulmonary tuberculosis.

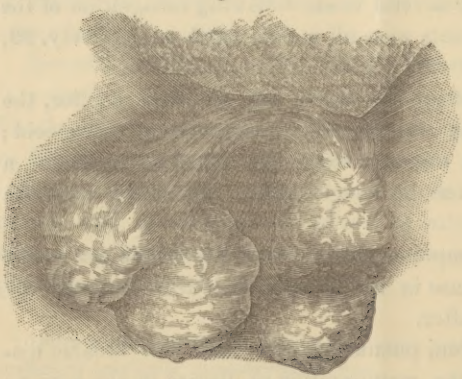
Case I.[1] (No. 17,250).—The first laryngeal lesion, recognized, was a shallow irregular ulcer on the left side of the posterior face of the pallid and thickened epiglottis. Ulceration soon attacked the right side also; then the central portion of the edge of the epiglottis, and subsequently its laryngeal face. Thus the epiglottis became encircled, as it were, with an ulcerating girdle, and gradually underwent destructive ulceration from above downward, till nothing but a hemorrhagic stump remained. At the autopsy it was found that the ulceration which had surrounded the epiglottis had extended into the base of the tongue, and had destroyed a portion of its substance. The ulceration on the laryngeal surface of the stump of the epiglottis was quite extensive, as was that also on the arytenoepiglottic folds and the ventricular bands. The vocal bands were intact, as was also the whole of the subglottic mucous membrane of the larynx and of the trachea, as far as it had been removed. This is distinctly shown in the specimen, herewith presented.

Dr. Seiler kindly made a number of sections of this larynx, two of which are now under the microscope for inspection. One shows small-celled infiltration and caseous degeneration in the stump of the epiglottis; and the other exhibits infiltration with cheesy centre in the mucous membrane, and infiltration in a mucous gland.

Tubercle was abundant in both lungs in various stages of degeneration. Several small cavities were seen in the upper lobe of the left lung; but there were none in the right lung.

Case II. (No. 21,110).—Geo. F., aged 29, a German, blonde, unmarried, and for fourteen years a seaman, had no record of sickness prior to six weeks before being sent to me for laryngoscopic examination. Exposed to very cold weather in the English Channel, he acquired what was probably an acute laryngitis, attended by dysphonia, dysphagia, cough and expectoration. The dysphagia increased until swallowing had become exceedingly difficult and exquisitely painful. His pain, indeed, was the principal subject of complaint.

Fig. 3.



Ulcerative acute tuberculous laryngitis.

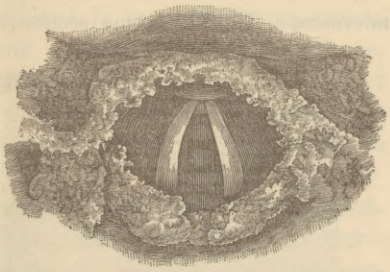
Nutrition seemed good. Lung capacity was of normal average. There was no sign of dyspnœa on exertion. There was slight dulness on percussion at the apex of the right lung; and bilateral exaggerated vocal resonance on auscultation posteriorly. The mucous membrane of the gums of the upper teeth was studded with tuberculous-looking elevations.

Laryngoscopy revealed (Fig. 3) almost complete ulcerative destruction of the

[1] For details with illustrations, see Archives of Laryngology, vol. ii. No. 2.

right half of the epiglottis. The ulcerations extended into the glosso-epiglottic sinuses in the one direction, and into the aryteno-epiglottic fold in the other; the glosso-epiglottic ligament and aryteno-epiglottic fold being destroyed in considerable extent. The ulceration extended into the base of the tongue on that side. A few red unhealthy granulations existed at the junction of the crest of the epiglottis with its left side; ulceration existing over the whole of that side of the epiglottis, also, but much less deeply than on the right side. The left edge of the epiglottis was several times the normal thickness; and a deep oval excavated ulcer occupied its free edge. There was immense tumefaction of both supra-arytenoid eminences. The left side of the larynx was completely hidden; and the interior of the right side indistinguishable. Progressive ulceration gradually destroyed so much of the swollen epiglottis and aryteno-epiglottic folds as to fully expose the interior of the larynx to inspection (Fig. 4), when it was seen that the vocal bands were intact; as had been inferred from the character of the voice.

Fig. 4.



Progressive ulceration in acute tuberculous laryngitis.

Post-mortem examination revealed complete tuberculous infiltration of the right lung, and almost equally extensive infiltration in the left lung; only a few cubic inches in the anterior portion of the lower lobe being free from the product. The lungs contained no vomicæ.

There was extensive ulceration of the base of the tongue, the remnant of the epiglottis, both aryteno-epiglottic folds, and both lateral laryngeal walls almost to the edge of the ventricular bands. No macroscopic lesions were apparent on the vocal bands, or in the subglottic portion of the larynx, or in the entire trachea, or in so much of the primitive bronchi as was removed with the specimen, which is herewith presented for inspection.

In both these cases the tuberculous lesions are limited to the supra-glottic portion of the larynx, as was likewise the case at the last laryngoscopic examination of Case III., the pathological particulars of which, I shall, probably, be prepared to present to the Pathological Society at no distant date.

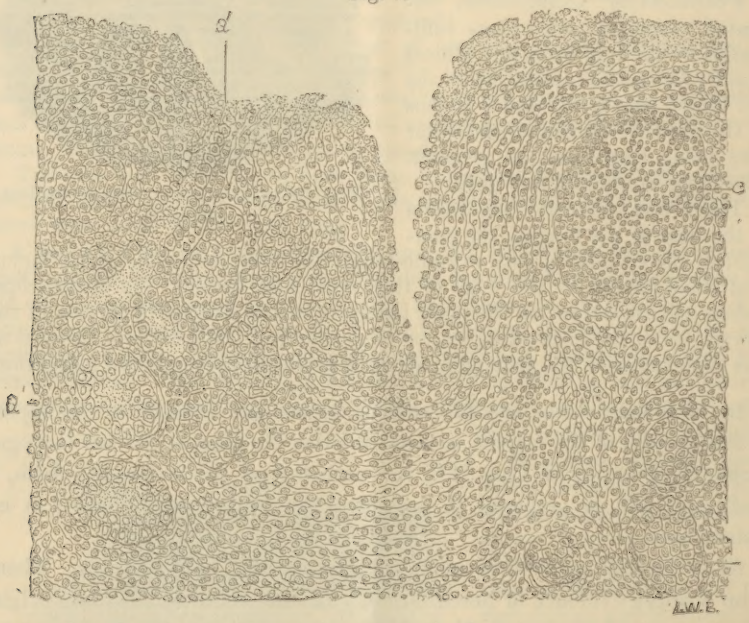
As to the etiology of these cases, we are restricted to hypotheses. There is no positive evidence of hereditation. In two, there was distinct origin in a severe cold, most probably an acute laryngitis.

In the third, there was no recollection of any special cold; but it is not improbable that the disease began as an acute or subacute laryngitis, or laryngo-bronchitis, milder in character than in the other cases. It is quite possible, further, that there may have been some slight pneumonitis accompanying the laryngitis or laryngo-bronchitis in these cases, and that the caseous foci of some of its residual products in the lung originated the tuberculization.

On the whole, therefore, I am inclined to the belief that cases of so-termed primary tuberculosis of the larynx may be relegated to the category of secondary tuberculosis, commencing very early and running an unusually acute course.

Secondary tuberculosis of the mucous membrane of the larynx presents us with two stages: (1) that of infiltration; and (2) that of ulceration; several specimens of both of which conditions are under the microscopes before you. No tubercle is found in the epithelium; the infiltration always taking place beneath the epithelium. The infiltration is found both in the mucosa and the submucosa; in the latter, however, rarely as deeply as the situation of the mucous glands; according to some, never; but this negation is too absolute, as will be proved by several sections now under the microscope (Fig. 5). One section, through a ventricular band, exhibits

Fig. 5.



Tuberculous ulceration; involvement of gland. Section through mucous membrane at base of epiglottis; *aa*, gland and its duct infiltrated with granular tubercle; *b*, acinus of gland; *c*, tubercle. × 180.

granulation tubercle extending more and more densely in the very vicinity of the glands, everywhere infiltrating the interacinal connective tissue, and in many places so profusely infiltrating the glands as to render it difficult to tell whether a given mass is a tubercle or an infiltrated gland. In some instances, as in some of the specimens before you, the infiltrate is uniformly

disseminated through the entire thickness of the mucous membrane; but in the great majority it is found only in the upper part of the mucosa, just beneath the epithelium. Sometimes there is quite a free space (Heinze) between the epithelium and the most superficial tubercles. The overlying epithelium appears normal; and remains well attached unless ulceration has actually begun. Individual tubercles are noticed more abundantly in the upper portions of the mucosa, and more and more sparsely towards the deeper. In these portions, too, there is less granular infiltration than there is above. In the sections exhibited, the older tubercles occupy the central portion of the mucous membrane chiefly; young ones, the sub-epithelial portion. Giant cells are few in number. Advanced cases exhibit extensive caseation, both in the tubercles and in the tissue immediately contiguous; especially near the periphery.

Miliary tubercle is beautifully exemplified in one of the preparations under the microscope, from the larynx of an infant seven months of age. The section, for which I am gratefully indebted to our accomplished curator, Dr. Seiler, includes the entire circumference of the larynx directly through the glottis, and the tubercles in the field of the instrument are located in the interarytenoid fold.

Circular infiltration occurs partly outside the adventitia of the blood-vessels; but also, and to a greater extent, imbedded between its fibres. Fully formed tubercles are sometimes observed; occasionally with evidence of central caseation. The lumen of the vessels is obliterated by pressure in many places. Extensive infiltration has destroyed parts of the adventitia; but the integrity of the remaining coats of arterial vessels is usually well maintained. So likewise with the capillaries; while the more delicate tunics of the veins readily undergo destruction; mere traces remaining in some localities.

As regards the glands, to ulceration of which a tuberculous character has been so much attributed both by many clinicians and not a few pathologists: while they are not directly involved in the tuberculization as a rule, they undergo, when implicated, two processes of infiltration simultaneously.

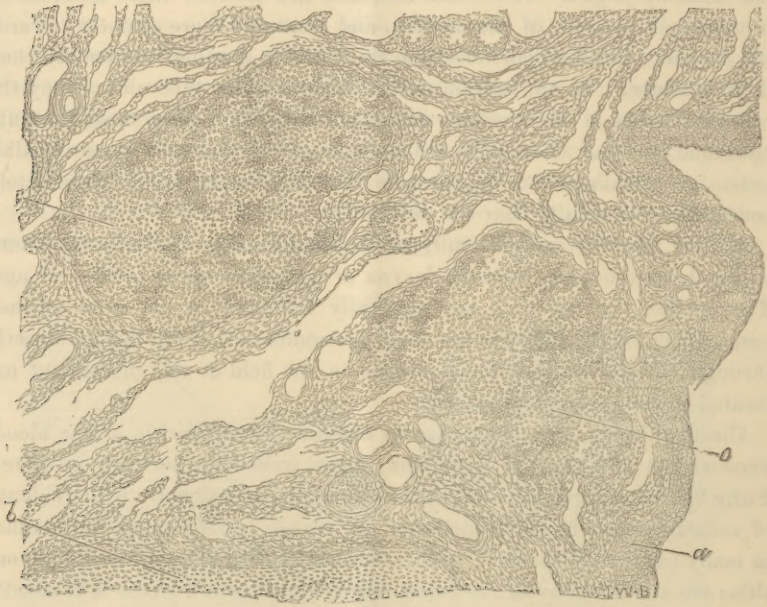
I. *Inter-acinous; i. e.*, great increase of round-cells in the interacinous connective tissue; or infiltration between the acini.

II. *Intra-acinous; i. e.*, interstitial increase of round-cells; infiltration within the acini.

The interacinous infiltrations separate the individual acini, and partially compress them. During this process, or somewhat later, miliary tubercles, likewise, collect both in the connective tissue between the individual glands, and between the individual acini of individual glands. The gland-cells lining the proper membrane become detached and undergo destruction; and, as the membrane is forced inward by the external pressure exerted upon it, the diameters of the acini show their compression from globular into irregular oval, and elliptic bodies. Many acini finally

undergo partial or complete destruction, as may be, from fatty degeneration following the combined internal and external pressure. The ducts (Heinze) resist the process longer than the acini.

Fig. 6.



Miliary tubercles in inter-arytenoid fold of an infant seven months old.[1] *a*, epithelium; *b*, right supra-arytenoid cartilage; *c*, tubercles.

Secondary tuberculosis of the larynx occurs, according to my own clinical observations, both in an acute and in a chronic form. The acute form occurs chiefly in cases of rapidly caseating pulmonary tuberculosis, is liable to occur quite early in the disease, and has an average life of from six to eighteen months. The several varieties of the more chronic forms occur chiefly in the more languid cases commencing as localized pneumonitis, occur at a comparatively advanced stage of the disease, and last from two to four years, or even longer.

The earliest recognizable stage of the *acute* form is almost always manifested by marked *congestion* of the mucous membrane. The earliest recognizable stage of the *chronic* and much more frequent form, is almost always manifested by marked *pallor* of the mucous membrane.

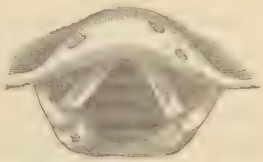
In the acute form.—The period of the pulmonary disease at which the secondary tuberculosis takes place is not uniform. In most instances,

[1] This specimen shows, too, the layer of squamous epithelium at the posterior wall of the larynx.

evidences of softening are indubitable when the laryngeal disease comes first under observation; but in many they are absent, or escape detection.

The intense catarrhal laryngitis of the acuter form usually subsides, in the course of from two to three weeks, into a severe chronic catarrhal laryngitis, indistinguishable, laryngoscopically, for a considerable period (perhaps as many as two to six weeks) as a malady due to specific constitutional disorder. In the course of from three to six weeks, a number of superficial ulcerations become noticeable upon the surface of the mucous membrane; most frequently upon the upper portion of the posterior surface of the epiglottis, frequently upon the inter-arytenoid fold, less frequently upon the inner face of the arytenoid or supra-arytenoid cartilages, upon the vocal bands, or upon the ventricular bands, and other portions of the interior of the larynx (Fig. 7). These initial multiple superficial ulcerations of quasi-tuberculous origin may not be followed or succeeded by any other local manifestations characteristic of tuberculosis.

Fig. 7.



Multiple initial superficial ulcerations.

They present at first the closest physical similitude to the catarrhal epithelial erosions or aphthous ulcerations of a chronic laryngitis with especially irritating secretory products. Suspicion as to their dependence upon tuberculosis is excited by their multiple manifestation; purely catarrhal erosions being solitary, or very few in number.

The shape presented by these superficial ulcerations differs with the locality they occupy. They are roundish or ovoidal on the epiglottis, and upon the pharyngeal surface of the supra-arytenoids; elliptic or linear on the vocal bands and on the inter-arytenoid fold. This difference in configuration, being unusual in mere catarrhal erosions, is indicative of a difference in character. Catarrhal laryngitis coexists with the condition under consideration, as in several other varieties of intra-laryngeal disease; but the multiple ulcerations seem specifically due to the underlying dyscrasia, and not to the attendant catarrh.

Erosions purely catarrhal in origin may also exist, but they are not essentially part and parcel of the tuberculous malady.

These individual ulcerations gradually extend in depth and in periphery, and often coalesce. Thus they can rarely be submitted, in their initial state, to the observation of the pathological anatomist.

Erosions similar to these, however, sometimes take place from time to time as fresh manifestations in the more chronic form of the affection; and these latter forms often come under post-mortem observation; as in the specimen herewith presented.

Inferentially it may be presumed that the early erosions in the acuter forms of laryngeal tuberculosis are similar to these in their histologic pathology.

The fresh superficial ulcerations last alluded to, exhibit under the microscope a loss of substance, confined to the epithelial layer in many examples, while extending in others to the immediately subepithelial portion of the mucosa also. Portions of epithelium implicated, but not yet exfoliated, are turbid; and, at some points, partially detached.

Ulcerations extending somewhat deeper into the mucosa show different stages of cell infiltration, erosion of vessels, accumulations of detritus and fatty degeneration. But *no miliary tubercle can be detected either in the beds and edges of the ulcers, or in the tissues in their immediate vicinity.*

The broken down débris of tubercle presenting nothing characteristic, the tuberculous nature of these initial superficial ulcerations is inferred, therefore, from the fact that they are rarely observed apart from subsequent undoubted tuberculous manifestations in different portions of the laryngeal mucous membrane. If this inference be justifiable, it should be admitted that tuberculous ulceration may occur independently of direct tuberculous infiltration *in situ;* in which case they might probably be accounted for by so great a constitutional proclivity to tuberculous degeneration as to favour ulceration as a result of local irritation of almost any kind. Should this view of the subject be unsatisfactory, we would be obliged to regard these ulcerations as non-tuberculous in character; or to acknowledge the probability of an actual infiltration with tubercle, and its rapid destruction and discharge. Later in the disease, when these ulcerations have extended in depth and in periphery, or by coalescence, their positive tuberculous character becomes manifest; as is evident, after death, by the detection of secondary miliary tuberculosis in parts immediately contiguous.

As previously intimated, it is both a matter of doubt and of dispute, whether these shallow ulcerations are preceded by miliary tubercle. They come to the notice of the laryngoscopist, as a rule, only after the disease has made such progress as to have rendered an examination almost a matter of necessity; and he misses the pre-existent lesion.

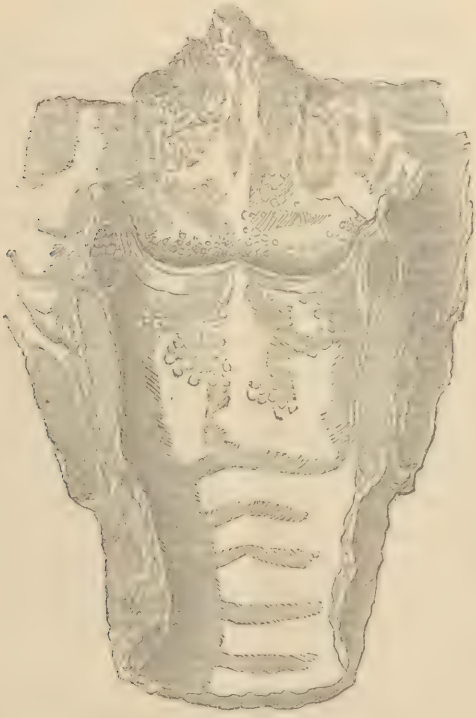
In this connection it will be useful to mention an instance in which Prof. Schnitzler of the Vienna University claims to have had an opportunity, and the only one he has had, to watch the development of shallow ulcerations from miliary tubercles.[1] It is the most useful instance that I have come across in my reading; although fully as authoritative assertions have been made by others as to having encountered cases in which they could detect the miliary tubercles laryngoscopically. These opportunities are so

[1] Zur Kenntniss der Miliartuberkulose des Kehlkopfes und des Rachens, Wien, 1881.

rare, however, that Tuerck never reported more than one in his famous clinical collection; and Stoerck, of Vienna, now the oldest living laryngologist, has reported but one from his extensive experience.

As for myself, I have never been able to detect tubercle laryngoscopically; not even in the case of the preparation which I present, in which ample evidence of tuberculous infiltration was visible macroscopically in the well washed specimen (Fig. 8).

Fig. 8.



Tuberculous infiltration of larynx.

To resume the clinical pathology of laryngeal tuberculosis, it is to be remarked that subsequent laryngeal manifestations of local tuberculosis may be moderate or severe. As a rule, some characteristic circumscribed tumefactions attract notice not many days after the advent of these shallow ulcerations, and while they are increasing in depth and in periphery and by coalescence. Similar characteristic changes are manifested in some instances previously to any ulceration of the mucous membrane whatever. They take place at almost any stage of the malady; and they may remain the sole visible manifestation of the tuberculosis. These circumscribed tumefactions are chiefly thickenings of tissue, and are observed at various points of the structures.

The intumescence occurs chiefly in the mucous membrane of the epiglottis, ventricular bands and vocal bands; and less often in the inter-arytenoid fold and aryteno-epiglottic folds; in the last named structures, perhaps, less frequently than in any others; presenting, in this respect, a marked contrast to the more chronic form of laryngeal tuberculosis, in which these selfsame aryteno-epiglottic folds are much more frequently involved than any of the other tissues.

The epiglottis undergoes tumefaction on its posterior aspect, to several times its normal thickness; its functions as an obturator to the larynx in deglutition being thereby impeded.

The ventricular bands thicken so as to overlap the vocal bands to a considerable extent; and even to conceal them from view in some instances.

The vocal bands become thickened into veritable *vocal cords* indeed; and their inferior aspect becomes tumefied; sometimes to such an extent, that, as in a specimen exhibited by me before the society,[1] some years ago, for the purpose of demonstrating the fact, they are transformed into thick, bulky, tumour-like folds, absolutely larger than the ventricular bands themselves.

When these tumefactions are multiple or bilateral, as often occurs, their encroachment upon the calibre of the larynx is in some instances so great as to produce a veritable stenosis, threatening asphyxia, and sometimes requiring tracheotomy.

Not only do these tumefactions occur with the ulcerations just discussed; but they also accompany other intra-laryngeal tuberculous processes. They may also take place without previous ulcerations or other visible manifestations of tuberculous disease.

The swollen mucous membrane becomes irregularly tumid, acquires a pallid yellowish-gray or gray colour, as is observed in the early stage of the more chronic form, looks sodden and corrugated, and often supports a dingy yellowish pultaceous deposit.

Under the microscope, these swellings are found to be due to copious infiltration of small (lymphoid) cells from immediately beneath the epithelium inwards, both in the mucosa and in the sub-mucous connective tissue; massed here and there into tubercle nodules or groups of miliary tubercle, some of them undergoing central caseation. They extend from the epithelial layer as far as the glandular structure; the older tubercles being usually the deeper-seated ones. The glands, as a while ago stated, are not often themselves infiltrated, but the infiltration is usually massed around them and between their individual acini, which are thus compressed out of shape and subjected to fatty degeneration from pressure.

When the epithelium becomes detached, hemorrhagic ulcerations are thus exposed, of undoubted tuberculous character. These ulcers rapidly

[1] Trans. Path. Society, Phila., vol. v. p. 83.

extend in the most irregular manner; so that large portions of tissue are soon included in their ravages. Their edges are well defined and often slightly hemorrhagic or injected; their beds are rough and irregularly mamellonated, and usually covered with caseous detritus. The ulcers deepen and deepen in convergent outlines; or undermine the surrounding tissue at different points of their periphery. They extend to the ultimate limits of the cell infiltration, and may thus lay bare the very perichondrium—the first structure that seems fashioned to resist actual tuberculization.

Submitted to the microscope these ulcerations usually present one of three conditions:—

I. No evidence of tubercle in either bed or edge of the ulcer, but infiltration of granular tubercle in immediate contiguity to the ulcer, or but a short distance from it;

II. Either infiltration of granular tubercle, or nodular tubercle in edges or bed, or both, without either contiguous or distant infiltration; and,

III. and most frequently, tubercle in the edges and bed of the ulcer, associated with granular tubercle-infiltration, whether contiguous only, or copiously disseminated throughout the mucous membrane.

As the disease progresses, nearly the whole interior of the larynx becomes involved. The tissues, generally, become so swollen that all the sharp outlines (edges of ary-epiglottic folds, vocal bands) become lost in thick welts. Later, as the ulceration extends, the whole structure becomes transformed into an irregular, excoriated, ulcerating, almost fungoid mass. Especially is this marked in the epiglottis and vocal bands; the latter, by fissure-like longitudinal ulcerations, becoming converted into a series of bands.

In many instances, local suppurative centres of inflammation become established in the immediate contiguity of these ulcerations, and, as they reach the perichondrium, involve that structure also, so that the cartilage becomes exposed.

The cartilage, too, becomes attacked, and undergoes destruction, in débris, as is most usual with the epiglottis (Fig. 9), or in fragments and in mass, as with the vocal processes, arytenoids and supra-arytenoids, and even the cricoid; or in large plates, as with the cricoid and thyroid cartilages. These fragments are often discharged by expectoration; the posterior vocal processes, and even the entire arytenoid cartilages, being, at times, exfoliated in mass. In the dead body, such fragments, or detached cartilages, are often seen loose in the abscess which surrounds them.

The epiglottis is sometimes destroyed in its entire free portion, by progressive ulceration from above downward, so that a mere deformed stump remains; occasionally the destruction is by progressive ulceration from the side.

The similitude of the laryngeal pictures of this variety of laryngeal

tuberculosis, to those presented in presumptive primary laryngeal tuberculosis, indicate to my mind such similitude in character that the latter may be regarded as acuter examples of the former. Both occur in cases

Fig. 9.

Tip of epiglottis, showing tuberculous ulceration and necrosis of cartilage, × 60. Section cut by Dr. Formad.

of rapid pulmonary tuberculosis; both become associated with secondary tuberculosis of the trachea, pharynx, palate, tongue, lips, and other structures; both progress without interruptions.

The chronic form of tuberculosis of the larynx is not ushered in by congestion following distinct history of exposure, as in the acuter form just described; but it is characterized by well-marked pallor of the mucous membrane; a pallor participated in, in many instances, by the mucous membrane of the pharynx and mouth. This anæmia is often apparent long in advance of similar evidence of impairment of nutrition elsewhere.

Although somewhat characteristic, and strongly suggestive of constitutional tuberculosis, this appearance is not peculiar to the tuberculous larynx only. In many cases, congestion ensues at a later period of the disease; as a result of the irritation produced by the tuberculous process; —but in many, the pallid hue continues to the very last.

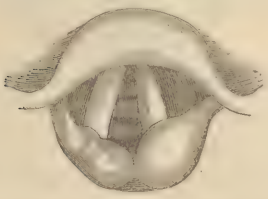
Accompanying this local anæmia, there is an irregular vascularity of portions of the mucous membrane, which, in localities where its connections are loose (inter-arytenoid fold, ventricular bands), is elevated here

and there in irregular wrinkled ridges or welts, red or gray in colour, often of a distinctly villous aspect.

Some instances present spots of ecchymoses, or little irregular varices, irregularly located on the laryngeal surface of the epiglottis, or on points in the interior of the larynx proper. There is often a general tumid condition of the mucous membrane; but this is by far a less frequent condition than circumscribed tumefactions of a peculiar character.

I. The most frequent locality of tumefaction is in the tissue inclosing the supra-arytenoid cartilages and the apices of the arytenoid cartilages; the least frequent point of intumescence in the acute variety. These tissues, for the intumescence is most frequently bilateral, undergo gradual transformation into pale, tumid, club-shaped, and finally distinctly pyriform or irregularly globose tumours, gradually tapering off, stem-like, toward the epiglottic extremity of the aryteno-epiglottic fold; with obliteration of all evidences of demarcation between the edge of the fold and the peculiar outline of the cartilages of Wrisberg and Santorini (Figs. 10, 11), and the intervening tissue.

Fig. 10.



Supra-arytenoid pyriform intumescence, at an early stage.

Fig. 11.

Supra-arytenoid pyriform intumescence at a later stage; with thickening in the epigottis likewise.

Although most frequently pale in colour, these pyriform tumefactions sometimes become very much injected; sometimes intensely so; in exceptional cases, to actual lividity at the points where they press against each other in the movements of phonation and deglutition.

The tumefaction, if unilateral, or the greater of the two, if bilateral, is, according to my own experience, almost always situated on that side of the body corresponding to the lung more advanced in disease. This peculiar pyriform aspect of the aryteno-epiglottic fold is sufficiently characteristic of coexistent pulmonary phthisis to establish the diagnosis. It occurs chiefly in slow cases, commencing with a localized pneumonitis; and is noticeable at a comparatively advanced period of the first stage of the disease.

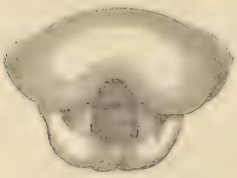
It is a hyperplasia due to extensive proliferation of lymphoid cells in the adenoid tissue normally very abundant in this structure.[1] The

[1] Wagner, Das tuberkelähnliche Lymphadenom. Leipzig, 1870-1.

increase of thickness of the mucous membrane is often three- or fourfold; so that it not infrequently measures fully one centimetre. Later in the disease, both granular and miliary tubercle are found. These swellings, as I have seen them, never subside, except in such partial measure as they may have been due to effusion of serum or other products, as a result of the inflammatory process, set up mechanically by pressure, or by irritation. They are distinct from the slight serous œdema which sometimes accompanies protracted subacute laryngitis, with which they are occasionally confounded.

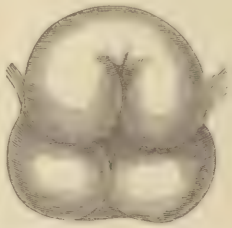
II. In another group of cases the epiglottis undergoes great thickening of its free surface, from tuberculous infiltration, into a tumid ridge of turban-shape (Fig. 12); its edge often presenting as a thick crescentic

Fig. 12.



Turban-like thickening of the epiglottis.

Fig. 13.



Crescentically swollen epiglottis overhanging the orifice of the larynx.

cushion or pad, in some instances overhanging the laryngeal orifice so as to conceal all the tissues from view, save, perhaps, the pyramidal intumescence of the supra-arytenoid cartilages (Fig. 13).

This may be associated with previously existing pyriform tumefaction of the supra-arytenoid and aryteno-epiglottic structures; or it may exist independently of any such manifestation at the time or at any subsequent period.

Ulceration usually begins superficially upon the laryngeal face near the edges, and gradually extends in depth and periphery. In most instances, progressive destruction takes place from above, until nothing but an irregular stump remains; but sometimes it proceeds from the side inwards, as admirably shown in the drawings exhibited.

As ulceration reaches the petiolus, it spreads along the commissure of the vocal bands into the ventricles, and along the ventricular bands.

Ulceration limited to the epiglottis is exceedingly infrequent, and is seen chiefly, if not only, in subjects who succumb rather early to the general malady. Ulceration of the anterior or lingual face of the epiglottis is unusual, and is almost always an extension of ulceration from the posterior face along the free edge. In one of the specimens under the microscope, however, the lingual face of the epiglottis is equally with the laryngeal face beset with tubercle infiltration.

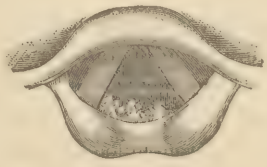
III. Still another group of cases may be classified in which the epiglottis is flaccid instead of rigid; and in which the peculiar manifestations begin on the posterior portion of the larynx, and chiefly on the inner surfaces of the meso-arytenoid fold—a point of tissue which almost invariably suffers likewise in cases which have begun with other manifestations.

This fold of tissue becomes red, its normal surface interrupted, irregularly tumid in projections which often increase in size until they simulate polypoid excrescences, condylomatous (Fig. 14) or accuminated (Fig. 15). Under the microscope, granular and nodular tubercles are found

Fig. 14.

Condylomatous inter-arytenoid infiltration; colour normal; softening right apex.

Fig. 15.



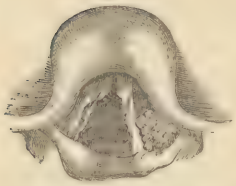
Accuminated inter-arytenoid infiltration; condensation right apex; red right vocal band; thick and irregular vocal bands.

beneath the epithelium. Small superficial ulcerations soon appear in this fold, or upon these projections of the mucous membrane. These ulcerations increase in periphery, coalesce, and become covered with yellowish, grayish detritus, which, when wiped off with a brush or sponge, or subjected to a douche or spray, reveal an irregular and slightly hemorrhagic surface; the ulcer having bled freely, though slightly, on contact with the cleansing substance. The surface is almost constantly covered with a thin layer of the products of secretion and disintegration.

In all these varieties of secondary tuberculosis of the larynx the normal colour of the vocal bands is retained in most cases. In some they are injected, sometimes deeply, and in occasional instances are hemorrhagic on some points of their surface. But in almost every case the peculiar polish of the surface, which presents such a remarkable appearance in health, is lost, and their aspect is dull.

In other cases, while the general surface of the vocal bands is congested, there are opalescent patches, dingy white in aspect, and more or less irregularly parallelogramic in configuration; probably groups of turbid pavement epithelia. Sometimes the smooth surface is interrupted by projections resembling excessive granulations. Ulceration takes place at the outer margins or edges of the vocal bands usually, and they become irregular in outline (Fig. 16); sometimes by small losses of substance, looking as though removed by a punch; sometimes in a jagged or more or less serrated edge.

Fig. 16.



Ulcerated vocal bands and ventricular bands, cavity left apex; solidification of right apex.

The breadth of the band is thus actually diminished at the points where ulceration exists, and often looks still more attenuated in consequence of the overhanging of the ventricular band. These ulcerations are most frequently seen at or towards the posterior extremities of the structures. Should the vocal band give way in great extent, as occasionally happens, retraction occurs in the fragments; and the shape of the glottis becomes, in consequence, very irregular. Should it give way at the posterior vocal process, that structure will be apt to project across the glottis.

Fungous vegetations are sometimes developed upon the ulcerated edges of the vocal bands; and these sometimes produce adhesions at the anterior portion of the bands.

Collections of tubercle within diffuse tuberculous infiltration is rarely observed in the mucous membrane of the vocal bands. It is infrequent also in the elastic fibrous tissue, in the muscular substance, or between the muscular fibres (Heinze).

IV. Still a series of cases may be differentiated in which the principal lesions are located in the vocal bands themselves.

The posterior surfaces of the vocal bands undergo great intumescence, so that they project beyond the vibrating edge of the band, and encroach seriously upon the calibre of the larynx. The dyspnœa from this stenotic condition is often sufficiently intense to threaten asphyxia, and only to be alleviated by tracheotomy.

Ulceration usually takes place in the longitudinal direction of the bands and the irregular appearance is quite suggestive of laryngeal neoplasms.

Tubercle is infrequent either in the muscular substance of the vocal band or between the muscular fibres.

Pyriform intumescence of the posterior portions of the aryteno-epiglottic folds has, in my practice, been far more frequent in young adults than in those in middle life; but I have seen the condition in patients as old as fifty-six.

Patients above fifty years of age are much more apt to exhibit, in my experience, torpid ulceration, with imperfect granulations, on the inner aspect of the mucous membrane over the supra-arytenoid and arytenoid cartilages; and most of the cases I have seen have exhibited both the ulceration and the pulmonary softening on the left side.

Perichondritis and chondritis, which usually attend the later stages of the malady in prolonged cases, are to be regarded as inflammatory processes of septic origin due to the tuberculosis; and not as tuberculous processes proper. Tubercle is rarely found close to the perichondrium; the infiltration, as has been stated, being chiefly in the upper part of the mucosa.

These processes may be limited in extent, be confined to one surface; or

they may be extensive and involve the entire surface, leading to proportionate destruction of tissue. But the processes themselves are by no means always proportionate to the intensity of the tuberculous process which has excited them.

Abscesses often result from the perichondritis, and usually point inwards towards the free surface of the larynx. They are liable to be productive of suffocative phenomena. Their incision and discharge, when practicable, usually relieves the threatening asphyxia, which, under other circumstances, may compel resort to tracheotomy.

After intra-laryngeal discharge of the abscess, spontaneous or artificial, fragments of cartilage are sometimes seen projecting into the free lumen of the larynx. This takes place most frequently with the posterior vocal processes, the arytenoid and supra-arytenoid cartilages, and much less frequently with the cricoid cartilage.

Tuberculous ulcers rarely heal. Exceptional cases of cure are occasionally noticed, but it becomes questionable whether those instances have been examples of actual tuberculous ulceration; and the conservation of the patient's life prevents an accurate answer to the query, for verification is possible only under microscopic inspection.

A disposition to heal is sometimes manifested by the appearance of healthy looking granulations upon the bed of the ulcer; but ere long the tuberculous infiltration occurs in the new-formed tissue which speedily succumbs to the invasion. Exuberant granulations are often developed in these ulcers, and even proliferate at times into veritable tumours or vegetations, which sometimes require removal to clear the air-passage from obstruction to respiration.

In cases of pulmonary tuberculosis following as a sequel to caseous pneumonitis, the general vitality of the tissues is impaired to such a degree that even follicular ulcerations of the larynx are insusceptible of cure.

The percentage of cases of pulmonary tuberculosis in which the larynx undergoes tuberculization has not been approximatively estimated; but it is probably not very large, and is much greater in hospital than in private practice. Heinze's personal pathological investigations yielded a percentage of 6.5. Men are more liable than women, in the proportion of nearly three to one; probably as the result of their greater exposure to inclement weather.

As mentioned earlier, the disease appears to be more frequently encountered in inherited than in acquired pulmonary tuberculosis.

The period at which tuberculization of the larynx occurs is variable, but as a rule the disease in the lungs is more advanced than in the larynx. If tuberculous ulceration has taken place in the larynx, cavities are almost always to be found in the lungs.

Inherited or acquired tendency to catarrhal inflammations of mucous membranes, especially to inflammations of the mucous membranes of the aerial tract, seems to be a causal factor in the development of tuberculosis

of the larnyx. Inheritance, in my experience, has seemed to be an important factor.

The tuberculous process itself seems to be an inflammatory process attended with profuse cell-proliferation, in tissues abundantly supplied with blood and lymph channels; the proliferation being too profuse for absorption, and undergoing maceration or decay.

Some acute observations of Dr. Formad appear to indicate an anatomical reason for the development of tubercle in certain subjects, and I trust that he will present these views to us this evening. Suffice it now to say, that he finds the lymph spaces extremely small in those animals most readily tuberculized, and likewise in the tuberculized subjects of the human species, and in tuberculous subjects only.

During the last three years I have, as opportunity permitted, examined the blood of many of my cases of tuberculosis of lungs and larynx. While often failing to encounter any appearance at all peculiar, I have so frequently observed a condition which has not been noticed in my examinations of blood in connection with other disease, that it seems to me to have some very close relation with tuberculosis. In all the cases with a temperature exceeding 100° F., this condition was almost sure to be found, and was rarely encountered at a lower body temperature.

In the first place, the proportionate number of white corpuscles is much diminished; secondly, the red corpuscles have a great tendency to become massed; and, thirdly and chiefly, plugs of granular matter become caught here and there between the individual members of certain groups of red corpuscles.

These granules look like granules which may have escaped from the white corpuscles, perhaps by dissolution of the protoplasm; and this of course would account for the paucity of the white corpuscles.

Clinicians have been too much inclined to regard the morbid manifestations which have been under consideration, as pathological processes remotely due to tuberculosis, but not in themselves tuberculous nor confined to tuberculosis.

Pathologists, with but few notable exceptions, view them as specific results of tubercle, the direct outcome of tuberculization, and not the mere result of irritation occurring in tuberculosis. The tenor of these remarks would indicate the greater confidence in the latter opinion.

NOTE.—Every point touched upon in the foregoing paper was amply illustrated by specimens, microscopic sections, and drawings. The various sections under the microscope were prepared by Drs. Longstreth, Seiler, and Formad, from three of the specimens exhibited; and they represented tubercle infiltration in the various component tissues of the larynx. The camera-lucida drawings, which are as beautiful as the preparations themselves, were made by Dr. Blackburn, under the immediate supervision of Dr. Formad. Without the assistance of the gentlemen named, it would have been impossible for the writer to have made his demonstrations so satisfactorily.

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